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ELECTROCARDIOGRAPHIC ANALYSIS OF
AURICULAR ARRHYTHMIAS

By P. M. Zlochevskiy

- USSR -

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ELECTROCARDIOGRAPHIC ANALYSIS OF AURICULAR ARRHYTHMIAS

Following is the translation of an article by P. N. Zlochevskiy entitled "Elektrokardiograficheskiy Analiz Mortsaatel'noy Aritmii" (English Version above) in Terapevticheskiy Arkhiv (Therapeutic Archives), Vol 32, No 5, Moscow 1960, pages 66-77.

(Reported at the meeting of the cardiological section of the Moscow Internal Medical Society 21 November 1959)

From the affiliate (Head--Professor B. B. Kogan) of the hospital internal medical clinic (Director--Professor A. L. Myasnikov Active Member of the Academy of Medical Sciences USSR) of the therapeutic faculty of the First Moscow Order of Lenin of the Medical Institute named I. M. Sechenov and the Fourth Chair of Internal Medicine (Head--Professor P. I. Yegorov Corresponding Member of the Academy of Medical Sciences USSR) of the Central Institute for the Advancement of Physicians

The 1950's were marked by a fundamental revision of the theoretical conceptions dealing with the mechanisms of auricular flutter and fibrillation. Previous concepts of a circus movement (Lewis and others) or of stage transmission of excitation (de Beer) have given way to the so-called "unitary" theory (Prinzmetal and others; Scherf and others), according to which the various changes in the rhythm of atrial origin depend on the occurrence of single or multiple heterotopic foci of impulse formation in the atria. Even previously, S. V. Andreyev, B. I. Borisova and V. S. Rusinov, observing the recovery of function of human hearts from cadavers, noted that in auricular fibrillation multiple foci of excitation

appear, and this confirmed Hering's hypothesis.

The electrocardiographic method occupies one of the chief places in the study of auricular arrhythmias. However, there is no agreement on the choice of the more demonstrative leads or the analysis of the atrial waves. The advantage of chest leads has been emphasized, particularly right-sided (G. Ya. Dekhtyar', M. V. Lashchovikov and others). G. Ya. Dekhtyar' recommends a lead from the right margin of the sternum in the third intercostal space (CR^{III}). L. M. Rakhlin, in addition to this lead (which he called "right atrial"), proposes a left-atrial CL lead in which the active electrode is located in the second intercostal space to the right of the sternum (CL^{II}). Various authors have noted the demonstrativeness of esophageal leads, bipolar chest atrial Lian leads, etc.

In almost all these studies, however, the considerable possibilities of comparative analysis have not been used, which give a synchronous demonstration of the leads under study, and no statistical treatment has been given. By means of four multichannel PTD-7 apparatuses designed by an experimental plant and the electrodynamocardiograph, "Elema" we were able to compare different variants of leads repeatedly and in different combinations. The type of auricular arrhythmia was determined by a classification proposed by E. A. Ozol. The size of the atrial waves has been made the basis of the classification: small waves under 0.09 mv, medium-sized waves of 0.1-0.19 mv, and large waves over 0.2 mv.

We distinguished a separate "zero" form (no atrial waves).

We gave ourselves the task of clarifying the selective area of atrial waves by means of CR leads, comparing the leads shown with other lead variants, figuring out the indications for selective leads and elucidating some problems of the origin of auricular arrhythmias according to EKG data.

At first, we studied the standard leads in 92 patients from 17 to 75 years of age among whom there were 35 men and 57 women. Forty-nine (53.2 percent) of the patients suffered from rheumatic involvement of the heart; 30 (32.6 percent) from arteriosclerotic myocardial fibrosis; 13, from thyrotoxicosis. In Table 1 the frequency of various types of auricular arrhythmias is shown (by the size of the atrial waves) in the standard leads. The advantage of the CR₁ lead is shown beyond doubt in the medium-sized and large-wave forms of auricular fibrillation at a frequency of 300-600 a minute. In 80 patients (87 percent) the atrial waves in this lead were relatively more pronounced. In only five cases were more distinct readings recorded in other leads (II, III, CR₂). The second division of Table 1 emphasizes the independence of the size of the atrial waves on the EKG of the frequency of the ventricular contractions.

The nature of the original disease made a definite impression on the appearance of the auricular arrhythmia. Our results are in complete agreement with the data of E. A. Ozol, according to which

chiefly large-wave and less often medium-sized wave forms of fibrillation are characteristic of the rheumatic lesions of the heart, while small-wave (Fig. 1) and less often, the medium-sized wave forms are characteristic of myocardial fibrosis (A. Tur also noted the predominance of large fibrillation waves in cardiac valve defects). In association with the tendency toward deviation of the electrical axis of the heart (more to the right in the rheumatic lesions and more to the left in myocardial fibrosis /from arteriosclerosis/), the evaluation of the size of the atrial waves can orient the physician as to the differential diagnosis. The possibility of combined arrhythmias should be noted, which we have usually encountered in patients with myocardial fibrosis. In one of them the auricular fibrillation was associated with a transitory left bundle branch block (see Fig. 1). In rheumatic fever, small-wave fibrillation was noted only three seriously ill patients with decompensation, myogenic dilatation of the heart and hydropericardium. E. A. Ozol found the same thing. In the majority of cases of thyrotoxicosis small and medium-sized wave forms of fibrillation were found.

Table 1

Atrial fibrillation in standard leads with different forms of ventricular arrhythmias and in the presence of different diseases

	Grassman						Engleheart						Hodson-Wade					
	I	II	III	CR ₁	CR ₂	CR ₃	CR ₄	CR ₅	CR ₆	CR ₇	CR ₈	CR ₉	CR ₁₀	CR ₁₁	CR ₁₂	CR ₁₃	CR ₁₄	
<i>All nonreentrant arrhythmias</i>																		
Hypertension	46	34	39	4	4	15	16	18	13	—	1	3	—	4	—	—	—	
Arteriosclerosis (0.1-0.6 mV)	55	42	35	26	45	54	48	15	5	5	16	11	3	3	14	6	6	
Arteriosclerosis (0.1-0.9 mV)	11	16	27	36	12	11	9	—	—	6	16	17	17	17	11	11	3	
Arteriosclerosis (>0.9 mV)	—	—	—	32	3	—	—	—	—	—	—	—	—	—	—	—	—	—
<i>Hyperthyroidism</i>																		
Hypothyroidism	92	92	92	72	72	91	76	35	16	26	48	49	20	13	—	—	—	
<i>Krone</i>																		
Hyperthyroidism	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Hypothyroidism	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

They: 1) type of atricular fibrillation; 2) leads; 3) form of ventricular arrhythmia; 4) nosologic forms; 5) slow; 6) medium; 7) fast; 8) rheumatic; 9) arteriosclerotic 10) thyrotoxicosis; 11) "zero"; 12) small-wave (under 0.09 mV); 13) medium-sized waves (0.1-0.15 mV); 14) large-wave (over 0.2 mV); 15) total; 16) number of pts.

The question arises, are all these leads recording the same qualitative process or does the successive recording of the leads reflect only transitions of various types of fibrillation or related arrhythmias, which fibrillation and flutter are? In practical electrocardiography the following conclusions are not uncommonly given: "atrial fibrillation and flutter", "transition of fibrillation to flutter", etc.

A synchronous examination made on 45 patients, in whom the readings of standard and left chest leads were compared with those of the right chest leads, can give the answer to this question. In Figs. 2 and 3 it is shown convincingly that here the same phase of a certain process was recorded in the atria, which was shown more prominently in the right chest lead: flutter or large-wave fibrillation. In only two cases, one of which is shown in Fig. 9, was it possible to record the transition of flutter into fibrillation within the limits of the same right chest lead.

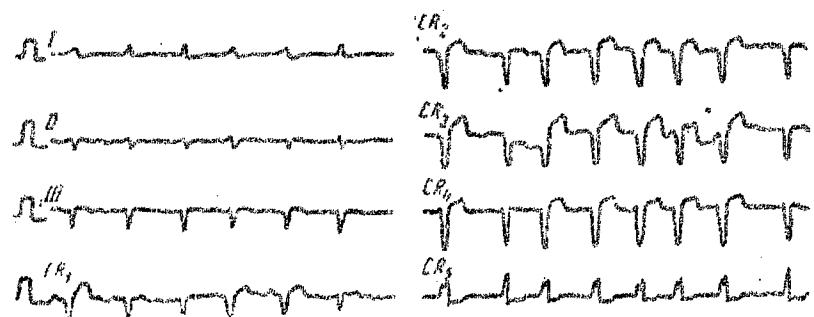


Fig. 1. Synchronous Recording of EKG of Patient T., age 65. Diagnosis: Hypertensive Disease Stage II, Arteriosclerotic Myocardial Fibrosis.

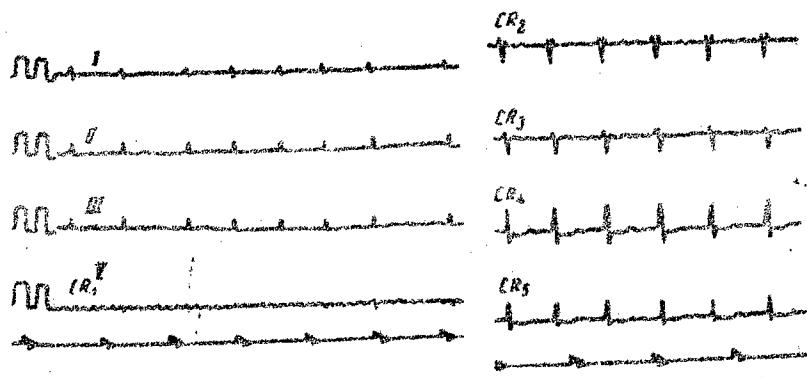


Fig. 2. Synchronous Recording of EKG of Patient G., age 66.
Diagnosis: Arteriosclerotic Myocardial Fibrosis.

Selective Recording of Atrial Flutter in CR^V Lead.

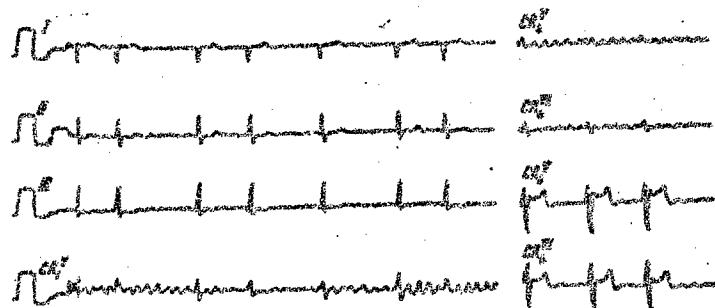


Fig. 3. Synchronous Recording of EKG of Patient T., age 20.
Diagnosis: Rheumatic Combined Mitral Valve Defect of the Heart

Chiefly large-wave fibrillation in CR^V; atrial waves in the leads from the right midclavicular line in the fifth and sixth inter-spaces are considerably larger than the waves taken from the corresponding sites on the left midclavicular line.

In 41 patients the electrocardiogram was recorded from different places on the anterior chest wall located at the second to eighth intercostal spaces on the main topographic lines.

In accordance with L. I. Fogel's nomenclature, which has been approved by the cardiological section of the Moscow Internal Medical Society, leads from the right half of the chest are denoted by the same letters, but before the figure designating the number of the vertical line a minus sign is written; on all the lines the appropriate interspace is designated with a Roman numeral superscript; this numeral is not used for standard leads.

In 50 patients the recording was made synchronously in the four leads (usually at the third-sixth interspaces on the lines mentioned). Therefore, a kind of topographic distribution chart of the "atrial potential" was constructed at various points on the anterior chest wall (Fig. 4). We subjected to statistical treatment the average numerical values of the maximum atrial waves, in millivolts in every lead (Table 2). In Table 2 the arithmetic mean values and their average errors are given.

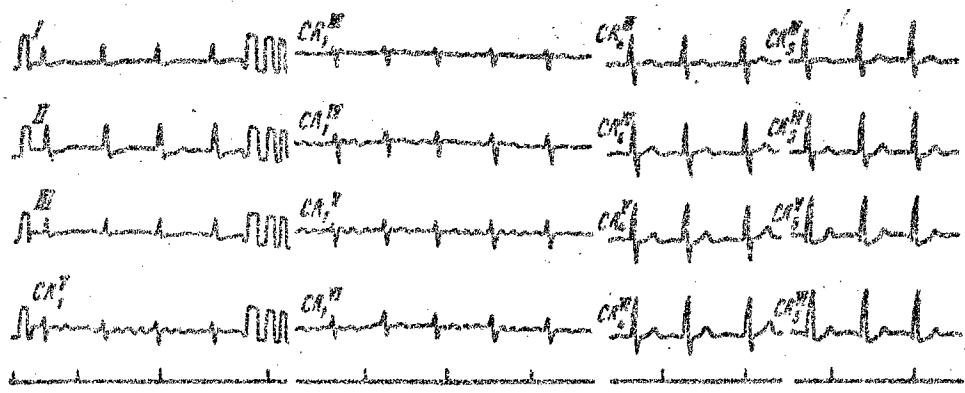


Fig. 4. Synchronous Recording of EKG of Patient OI, age 52. Diagnosis: combined rheumatic mitral valve defect of the heart.

Leads from different places on the anterior chest wall in the third to sixth interspaces. Only 15 out of 52 leads are shown. The most distinct atrial waves are in CII.

Table 2

Average Values¹ of Atrial Waves in Millivolts
Leads along vertical planes

Leads on the horizontal (interspaces)	CR_{-1}	CR_{-2}	CR_{-3}	CR_1	CR_2	CR_3	CR_4
Second	—	0.043 ± 0.011	—	0.047 ± 0.008	—	0.02 ± 0.003	—
Third	0.04 ± 0.01	0.031 ± 0.011	0.031 ± 0.01	0.034 ± 0.01	0.031 ± 0.013	0.035 ± 0.005	0.026 ± 0.009
Fourth	0.039 ± 0.012	0.038 ± 0.012	0.031 ± 0.008	0.038 ± 0.01	0.074 ± 0.014	0.04 ± 0.006	0.041 ± 0.008
Fifth	0.059 ± 0.011	0.092 ± 0.013	0.118 ± 0.01	0.118 ± 0.01	0.07 ± 0.014	0.036 ± 0.004	0.023 ± 0.006
Sixth	0.061 ± 0.013	0.079 ± 0.013	0.081 ± 0.012	0.094 ± 0.011	0.07 ± 0.013	0.04 ± 0.004	0.021 ± 0.006
Seventh	0.055 ± 0.008	0.056 ± 0.011	—	0.09 ± 0.012	—	0.03 ± 0.003	—
Eighth	—	0.03 ± 0.006	—	0.057 ± 0.012	—	—	—

¹The average errors are noted in the Table by a \pm sign.

The considerable variations in the average error are explained by the pronounced tendency of the indices to vary (from the zero to large-wave forms) with a relatively small number of observations. The difference of the average values of the atrial waves in leads CR_1 and CR_4^{III} exceeds the square root of the sum of the squares of their average errors by 2.4 times. According to Fisher's table, with the given number of observations the probability amounts to 0.02 and attests to the reliability of the difference obtained. The differences between the average values in the CR_1 and CR_4^{III} leads and in the CR_1^V and CR_4 leads do not satisfy this rule. However, in calculating the average difference between the maximum atrial waves in these leads we obtained 0.02 $\text{mV} \pm 0.007$ (probability of 0.01).

and 0.043 \approx 0.01 (probability of 0.001) respectively, which is statistically very reliable.

The left chest leads show considerably less distinct readings, not only in comparison with CR₁ but also with the symmetrically opposite distal leads on the right, for example, CR₄ (see Fig. 3).

Therefore, we determined the fact that in auricular arrhythmias the predominant distribution of the "atrial potential" is in the right half of the anterior chest wall, chiefly in the area of the fourth-sixth interspaces at the midclavicular line. Particularly distinct tracings are obtained in the CR₁^V (see Fig. 4), CR₂ and CR₃^V leads.

Parallel studies which we made in 1955 in patients with chronic cor pulmonale also showed that the area of the most pronounced so-called "pulmonary P waves" has the same distribution. In connection with this, these points, particularly CR₁^V, can be used as selective ones for the purpose of recording the potential of the right atrium chiefly. This is accounted for by the proximity of the right atrium to the selective leads, and in the case of auricular fibrillation apparently also by the location of the principal ectopic focus, which, according to the data of Prinzmetal and others, is usually located very low in the atria.

In four patients with atrial flutter and a constant factor of incomplete conduction of atrial impulses to the ventricles (terminology after A. N. Sigal) the advantage of the right chest

lead, CR₁, was also determined through synchronous recording of the electrocardiogram. Through two examples we were able to confirm the principle emphasized by A. L. Nyaamikov that the ventricular arrhythmia in atrial flutter is determined chiefly by the variations in atrioventricular conduction. The reflex effect on the atrioventricular conduction through the vagus nerve from the inhalation test (Fig. 5) and through pressure on the right carotid sinus area (Fig. 6) produced an excitation arrhythmia of the ventricles. Thereby the shape and frequency of the atrial waves did not change.

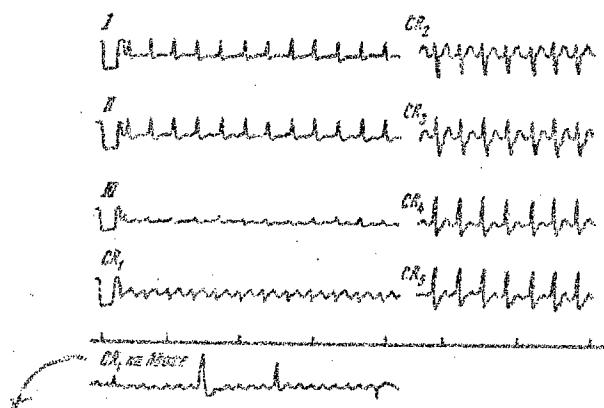


Fig. 5. Synchronous Recording of ECG of Patient T., Age 45.
Diagnosis: second-degree struma, thyrotoxicosis.

Atrial flutter with the appearance of a ventricular arrhythmia in CR₁ on inspiration.

The next section of the work was a study of the selective CR leads compared with other lead variants during synchronous recording. In all chest lead variants (CR, CP, CL and V) the

most distinct atrial waves were usually noted in the fifth interspace to the right of the sternum, particularly in CR₁^V, in comparison with which L. N. Rechlin's atrial leads were less demonstrative (Fig. 7). This applies particularly to the left atrial lead, CR₁^{II}, the average value of the atrial waves in which amounted to 0.04 mv ± 0.006 and were considerably lower than the readings in CR₁^V and CR₁ (see Table 2). Out of 20 patients larger atrial waves were recorded in the right atrial lead (CR₁^{III}) in only two with predominant mitral stenosis. This may be explained (in accordance with the experimental data of Prinzmetal and his associates) by the location of the principal ectopic focus in the upper part of the atrium.



Fig. 6. Synchronous Recording of EKG of Patient D., Age 58.
Diagnosis: pulmonary fibrosis with bronchiectasis; left-sided fibrothorax; cor pulmonale; arteriosclerotic myocardial fibrosis.

Atrial flutter. Slowing and excitation arrhythmia of the ventricles from pressure on the right carotid sinus area (noted by an arrow).

A. M. Sigal and A. I. Popova drew attention to the so-called "bipolar chest atrial leads" of Lian. In recording them the passive electrode is applied to the manubrium of the sternum, and

the active electrode is placed at the right of the sternum in the fourth (S_4) or fifth (S_5) interspace. The S_5 lead is considered more demonstrative. In the work of Lian and his associates the advantage of the S_5 lead over V_1 was shown for synchronous recording. The same thing was noted by A. I. Popova, who used a serial tracing. However, the method of the comparison given in these works suffers from inaccuracy, because the active electrode is located at different levels (in the fifth and fourth interspaces respectively). We made a synchronous recording with the Lian and CR_1 leads in 20 patients. Herby, the S_4 and S_5 leads were compared with the corresponding CR_1 and CR_1^V leads and showed the same picture in the majority of cases (fig. 8). In five cases somewhat more pronounced waves were shown in CR_1^V (CR_1), and in two cases, on the other hand, in S_5 (S_4). Therefore, the possibilities of these two lead variants are approximately equivalent. The greater demonstrativeness of S_5 was confirmed compared with S_4 . This once again shows that is not so much the method of using the leads as the location of the active electrode which is important.

Communications concerning the value of esophageal leads for fibrillation and flutter are not uniform in their content. Nyboer and Hamilton, M. Z. Neyman, who showed the advantage of esophageal leads, used serial EKG tracings and did not make any comparative analysis with right chest leads. Kistin and Bruce used the synchronous recording of esophageal and right chest leads but

frequently selected the CR₂ rather than the CR₁ lead, placing the chest electrode in the fourth rather than fifth interspace. These authors, like Butterworth and Poindexter, consider esophageal leads more demonstrative.

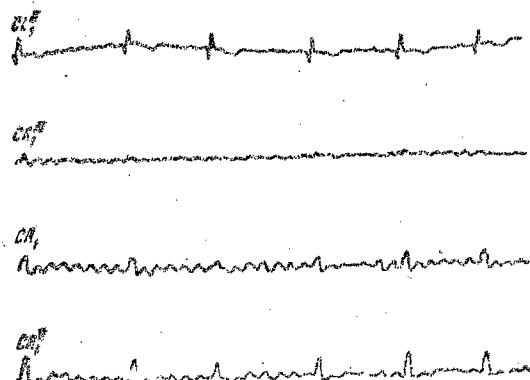


Fig. 7. Synchronous Recording of EKG of Patient K., Age 38.
Diagnosis: Combined rheumatic mitral valve defect with predominance of stenosis.

Showing a distinct advantage of the CR₁ and CR₂ leads over the atrial C1^V and C2^V leads (upper two curves).

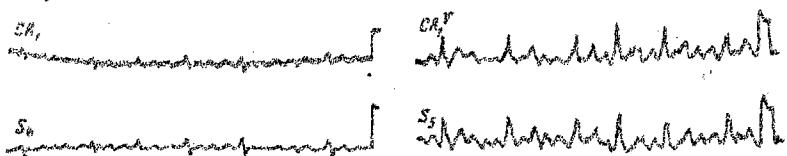


Fig. 8. Synchronous Recording of EKG of Patient H., Age 33 (on the left) and of Patient K., Age 30 (on the right) with a Rheumatic Combined Mitral Valve Defect in the Right Chest and Bipolar (after Lian) Leads from Analogous Interspaces

We recorded the esophageal lead synchronously with the CR₁^V lead in seven patients. In four patients the electrode hook-up

was the usual one: active electrode from the left arm to the esophageal catheter; passive electrode, to the right arm. In three patients the reverse arrangement of the electrodes was used, proposed by V. G. Popov and V. I. Maslyuk. A synchronous tracing showed more distinct atrial waves in CR₁ in four patients (Fig. 9) and the same picture in two (Fig. 10). Larger waves were seen in the esophageal lead at the atrial level in only one patient. We are inclined to agree with E. A. Ozol that the esophageal leads do not have any particular advantages.

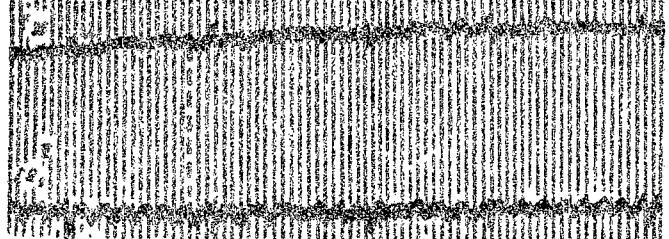


Fig. 9. Synchronous Recording of EKG of Patient S., Age 36, with Esophageal Lead (Eg₅) (with Reverse Arrangement of Electrodes) and CR₁, which was More Demonstrative. In CR₁ transitions of flutter into large-wave fibrillation are seen. Diagnosis: combined rheumatic mitral valve defect with predominance of stenosis.

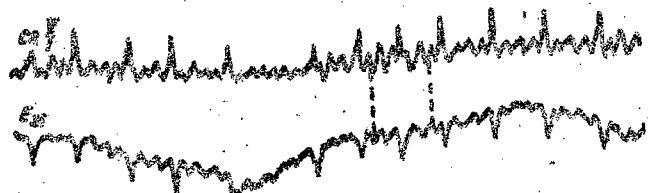


Fig. 10. Synchronous Recording of EKG with CR₁ and Eg₅ Leads in Patient K., Age 30. Diagnosis: rheumatic combined mitral valve defect with predominance of stenosis.

Showing the same pictures of the atrial waves. The peaks of the waves in Eg₅ coincide with the troughs of the waves in CR₁ and vice versa.

In the great majority of cases (86 out of 92) the large or medium-sized waves, readily seen and recorded in the selective leads

could not be considered auricular flutter, because they were different in shape and frequently arrhythmic. What term shall we use to define this type of atrial arrhythmia? In the works of S. V. Shestakov the old designation of Lewis is mentioned, "indistinct flutter". Ivan and others and S. V. Shestakov suggest considering these forms transitional between fibrillation and flutter. In our opinion, such an electrocardiogram shows atrial fibrillation with two varieties of waves—larger and slower, which arise at some single ectopic focus, and smaller and faster waves, which arise at multiple excitation foci; here, the activity of a single heterotopic focus is predominant. This may be confirmed by the synchronous recording of esophageal leads at the level of the left atrium and a CR₁^V lead, located over the right atrium. In four cases the recording of the esophageal leads by the usual method (the esophageal electrode is attached to the positive pole of a galvanometer) in synchrony with the CR₁^V lead showed opposite directions of the atrial waves. The peak in the CR₁^V lead coincided with the trough in the esophageal atrial lead and vice versa (see Fig. 10).

In making tracings by the method of V. G. Popov and V. I. Maslyuk, in which the esophageal electrode is hooked up to the negative pole of the galvanometer (three cases), a coincidence of the atrial wave peaks was noted (see Fig. 9). Since this method gives an upside-down tracing, it may be considered proved

that in all our seven cases, with synchronous recording of the leads mentioned located, as it were, over both atria, the directions of the large atrial waves were opposite. This permits us to suggest that the large atrial waves reflect a certain vector directed with its head end toward one of the atria or toward one of the atrial walls (anterior or posterior). It may be considered that such a vector is formed as the result of excitation of a solitary heterotopic focus, because the total vector, formed through the excitation of several foci, would have an altered direction, as a result of which the various phases of the atrial waves would coincide.

A comparison of the small waves on the synchronous tracings does not show any strict rules and regulations. Prinzmetal and his coworkers believe that fibrillation, like flutter, can start from just a single ectopic focus usually located in the lower part of the atrium and elaborating an excessive number of impulses. However, Scherf, Schaffer and Blumenfeld in their experiments produced not only a single-focus (application of acanthine) but also a multiple-focus (with faradization and acetylcholine) fibrillation and believe that fibrillation cannot be classified according to the number of ectopic centers. We believe that in electrocardiography such a classification may be based on the size of the waves, a criterion used by Soviet authors. The term "large-wave" (or chiefly unifocal) fibrillation should replace

the less suitable names "indistinct flutter", "fibrillation-flutter", "transitional form between fibrillation and flutter", etc. The larger the fibrillation waves the smaller are the foci of heterotopic excitation in the atria. With the increase in the number of ectopic foci, medium-sized and small-wave and even "zero" forms of fibrillation are recorded.

Another variety of atrial arrhythmia--atrial flutter with an inconstant (constant) factor of impulse conduction to the ventricles--should be diagnosed, as emphasized by A. M. Sigal, when there is strict constancy in the pattern of atrial waves over relatively long segments of the electrocardiogram.

Predominantly large-wave fibrillation in rheumatic lesions of the heart and the chiefly small-wave fibrillation in arteriosclerotic myocardial fibrosis can be explained by the nature of distribution of the process: more focal ectopic foci with a limited number of foci formed in rheumatic fever and diffuse with multiple ectopic foci in myocardial fibrosis.

Our investigations once again emphasize the fact that in the study of atrial arrhythmias the electrocardiographic method is one of the most important.

Conclusions

1. A selective area of more pronounced atrial waves in atrial fibrillation is located in the right half of the anterior chest wall in the fourth to sixth interspaces along the midclavicular

line. Particularly distinct tracings are recorded in the CR₁^V, CR₁, and CR₋₃^V leads.

2. With synchronous recording the selective lead CR₁^V is more demonstrative than Balklin's atrial leads and gives the same or more distinct results than the Lian bipolar chest atrial leads or esophageal atrial leads.

3. Large-wave auricular fibrillation is more characteristic of rheumatic hearts; the small-wave form, of arteriosclerotic myocardial fibrosis.

4. In practical electrocardiography the distinction of various types of atrial fibrillation by the size of the waves is expedient: large-wave, medium-sized-wave, small-wave and "zero" forms. The latter apparently reflect a fibrillation mechanism occurring as the result of activity of one, several or multiple heterotopic foci in the atria.

5. The existence of multifocal fibrillation, which has been established in experimental investigations, has been confirmed by synchronous recording of CR₁^V and esophageal leads at the atrial level, showing the existence of a definite vector directed toward one of the atria or one of the atrial walls (anterior or posterior).

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